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● LONG-ACTING SOMATOSTATIN ANALOGUE IN THE PREVENTION OF THE PANCREATITIS-LIKE CHANGES FOLLOWING ERCP

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The effectivness of the long-acting somatostatin analogue on the elevation of pancreatic enzymes after endoscopic retrograde cholangio-pancreatography (ERCP) was studied in a prospective, randomized double blind trial.

Patients and methods: Sixty-three consecutive patients undergoing ERCP were randomly allocated in two groups. In the control group 34 patients received isotonic sodium chloride, in the treated group 29 patients were given long acting somatostatin analogue (Sandostatin^{R}, Sandoz) 0.1 mg subcutaneously before the procedure. In all patients the same contrast medium (diatroziate, Uromiro^{R}, Bracco) was used. Blood was collected before and in predetermined periods after the pancreatography for measurement of amylase and lipase.

Results: No patients in the study developed clinical evidence of acute pancreatitis. The amount of contrast medium injected into the pancreatic duct did not differ significantly between the two groups. After endoscopy the amylase levels in the serum increased in 15 patients in the controls, and in 3 treated patients, while the lipase levels showed a rise in 17 cases in the controls and in 5 patients in the treated group. The difference is statistically significant between the groups concerning the enzyme changes (chi-square-test: amylase: 7.39, lipase: 8.75, $p < 0.01$). A significant difference could be observed in the amylase and lipase changes between the two groups at 90 minutes after ERCP. The enzyme levels showed only marked elevation in the treated group, however their rises were significant ($p < 0.05$) in the controls.

Conclusion: These findings suggest that the use of long-acting somatostatin analogue ameliorates the enzyme increases in the serum after ERCP. Therefore this compound might be of value in the prevention of pancreatitis-like changes and the pancreatitis associated with endoscopic retrograde pancreatography.

● SERUM NECROSIS FACTORS AND IMAGING PROCEDURES: COMPETITIVE OR COMPLEMENTARY FACTORS IN SEVERE ACUTE PANCREATITIS ?

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Contrast-enhanced CT scanning represents the gold standard for the staging of acute pancreatitis. In former studies we have demonstrated a high accuracy of C-reactive protein and antiproteases to detect pancreatic necrosis. The aim of this study was to find out the role of serum necrosis markers in comparison to CT scanning in the clinical management of severe acute pancreatitis.
Patients: 129 pats. with acute pancreatitis (male 87, female 42) entered the study. The mean age was 52.4 years (range 21-82). The etiology was alcohol overindulgence in 65 (50%), gallstones in 36 (28%) and other causes in 28 patients (22%). According to our classification (imaging, intraoperative findings, histology), 83 (64%) and 46 (36%) pats. suffered from interstitial edematous (AIP) and necrotizing pancreatitis (NP), respectively. Mean Ranson score was 1.4 and 4.7 in AIP and NP, respectively.
Methods: For two weeks after the onset of acute pancreatitis we analysed daily serum CRP, α-2-macroglobulin, α-1-antitrypsin, LDH and PMN-elastase. In addition we performed CT and ultrasound twice weekly. The data underwent computerized analysis and the overall detection rate (accuracy) for pancreatic necrosis was determined.
Results: Within 5 days after start of acute pancreatitis the overall detection rates for necrotizing pancreatitis were 86%, 84%, 82%, 72% and 69% using serum CRP (cut off 120 mg/l), PMN-elastase (120 µg/l), LDH (270 U/L), α-2-macroglobulin (1.5 g/l), and α-1-antitrypsin (3.5g/l), respectively. In comparison contrast-enhanced CT scan (gold standard intraoperative findings) reached 88% and ultrasound was beated off with 38%.
Conclusions: Because there are highly qualified serum markers which indicate necrotizing pancreatitis, such as CRP, PMN- elastase or LDH, a stepwise approach seems reasonable to stage pats. with acute pancreatitis: 1) Use serum necrosis markers. 2) If they indicate necrotizing pancreatitis use contrast enhanced CT scanning.

INFLUENCE OF ADVANCED AGE AND PREEXISTING DISEASES IN ACUTE PANCREATITIS (AP).

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The aim of this study is to establish if advanced age and some preexisting diseases negatively influence the severity and evolution of AP.

PATIENTS AND METHODS: 205 consecutive cases of AP (Jan 84-Apr 89). Study A: Subdivision of patients by age groups: <30 years (a); 31-50 years (b); 51-70 years (c); >70 years (d). Subdivision of patients by groups in relation to preexisting relevant diseases: arterial hypertension (ah); cerebrovascular and heart disease (chd); diabetes mellitus (dm); liver cirrhosis (lc); and in one group without preexisting diseases (wpd). Study B: A subdivision within each group was made, only of those patients strictly affected by biliary AP (n=154)

Determination of mortality rate, prevalence of severe morphological forms and complicated forms, length of hospitalization. Statistical evaluation of data by CHI square-test with Yates correction and Student t-test (length of hospitalization).

RESULTS: Only the following comparisons were found to be of statistical significance:

	Study A	Study B
mortality rate	lc>wpd	
severe morphological forms	b>d;c>d;wpd>dm	
complicated forms	wpd>ah	wpd>dm
length of hospitalization		c>d;wpd>ah

CONCLUSIONS: Our data suggests that advanced age and preexisting diseases considered in this study, with the exception of liver cirrhosis, are not determinant for considering the severity and evolution of AP. These two factors are strictly linked aboveall to complex pathogenetic mechanisms typical of AP.

● DIFFERENTIAL EFFECT OF PANCREASTATIN ON EXOCRINE AND ENDOCRINE PANCREAS IN VITRO

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Pancreastatin, a peptide isolated from porcine pancreas by Tatemoto et al. in 1986, is a potent inhibitor of insulin release and has also been shown to inhibit exocrine pancreatic secretion in vivo. The aim of this study was to investigate its direct effect on exocrine and endocrine pancreatic secretion and its indirect effect on exocrine pancreas mediated by the insulo-acinar axis.

Methods: The arterially perfused isolated pancreas of male Wistar rats was used. The exocrine pancreas was stimulated submaximally by CCK-8 (50 pg/ml perfusate). Insulin release was stimulated by glucose (15.8 mM). The pancreatic duct was cannulated by a polyethylene tube. Volume of and amylase content in 10 minute samples of pancreatic juice and insulin concentration in the effluent were measured simultaneously. Pancreastatin was tested in concentrations of 0.1, 1 and 10 ng/ml perfusate in 10 minute intervals ($n=6$; $p<0.05$).

Results: Insulin output increased with time of perfusion. 0.1, 1 and 10 ng pancreastatin/ml perfusate decreased insulin output by 44, 55 and 69% (2935±1316 vs. 1650±959; 4947±1968 vs. 2259±1619 and 10478±3529 vs. 3271±1872 µU/10 min). Volume of exocrine pancreatic juice and amylase output were stimulated by CCK (2.7±1.3 vs. 13.5±4.1 µl/1o min. and 4.1±2,7 vs. 109±31 U/10 min., respectively). Pancreastatin did not inhibit CCK-stimulated volume of or amylase output in pancreatic juice (volume: 13.5±4.1 vs. 15.5±5.2 / 12.6±5.6 / 13.4±6.0 µl/10 min; amylase output: 109±31 vs. 128±42 / 122±42 / 117±46 U/10 min., respectively).

Conclusion: Pancreastatin, in concentrations between 0.1 and 10 ng/ml perfusate has no effect on exocrine rat pancreas in vitro. Inhibition of insulin release by pancreastatin does not affect CCK-stimulated pancreatic secretion in vitro.